Charcot’s motor brain map and 19th-century neurosurgery

Richard Leblanc, MD, FRCSC
Montreal Neurological Institute and McGill University, Montréal, Québec, Canada

Neurosurgery is predicated on the knowledge of the structure-function relationship of the brain. When the topic is broached in its historiography, it begins with Fritch and Hitzig’s report on the localization of motor function in the cortex of the dog and skips rapidly to Wilder Penfield’s homunculus. In that gap are found the origins of modern neurosurgery in 3 papers published by Jean-Martin Charcot and Albert Pitres between 1877 and 1879 in which they describe the somatotopic organization of the human motor cortex and draw the first human brain map. Their findings, obtained through the clinicopathological method, gave relevance to David Ferrier’s observations in animals. Their work was extensively cited, and their illustrations reproduced by Ferrier in his landmark lecture to the Royal College of Physicians in 1878. It was known to William Macewen, who used localization to guide him in resecting intracranial mass lesions, and to William Osler and John Hughlings Jackson, who were early advocates of intracranial surgery. This paper describes Charcot and Pitres’ discovery of the cortical origin of human voluntary movement and its somatotopic organization, and their influence on 19th-century intracranial surgery. It fills a gap in the historiography of cerebral localization and neurosurgery.

https://thejns.org/doi/abs/10.3171/2020.10.JNS202651

**KEYWORDS** Charcot; history; localization; motor cortex; neurosurgery; Pitres; somatotopy

From antiquity to the latter half of the 19th century, cranial surgery was restricted to the elevation of depressed skull fractures, removal of bone shards, and trephination of the epidural space for a suspected blood clot. The development of neurosurgery as a specialized discipline was hampered by a lack of knowledge of the functional anatomy of the human brain. Functional localization was first achieved for speech by Pierre Paul Broca during a debate that raged in French academic circles between 1861 and 1865, and for human motor function by Jean-Martin Charcot and his student Albert Pitres in a more serene atmosphere, through 3 papers published between 1877 and 1879 and 2 books published in 1883 and 1895. This paper describes Charcot and Pitres’ discovery of the human cortical motor zone and its somatotopic organization, and argues that they played a determining role in the origins of 19th-century neurosurgery.

**Methods**
Charcot and Pitres’ publications on motor localization, and those of others written in French on the same topic, have been consulted and pertinent passages translated into English for the first time by the author. From these sources, and from the contemporaneous British literature, it is argued that Charcot and Pitres produced the first human brain map derived from postmortem examinations, and that they influenced the emergence of early intracranial surgery.

**The Standard Model of Motor Function Before Fritch and Hitzig**
The first experimental studies to demonstrate that different parts of the brain control the movements of different parts of the body were performed by Nicolas Saucerote, a surgeon in the French Army, in 1769. Saucerote observed that deep, penetrating lesions in front of the coronal suture produced paralysis of the opposite hindlimb of the dog, that lesions behind the coronal suture caused paralysis of the opposite forelimb, and that deeper lesions converging on the posterior aspect of the corpus striatum paralyzed the opposite side of the mouth. From these observations, Saucerote inferred that paralysis of the face, arm, and leg each resulted from damage to a specific part of the deep gray nuclei.

Achille-Louis Foville, the early 19th century’s great anatomist, found that Saucerote’s observations also applied to the human brain. In a series of clinicopathological studies starting in 1821, Foville observed that damage to the basal ganglia (probably in cases of hypertensive hemorrhage) could produce paralysis even if the cortex re-
mained intact. Thus, he concluded that the cortex was not essential to motor function but that the deep gray nuclei were:

When the arm alone is paralyzed...the cerebral alteration occupies the optic layer [the thalamus] and its projections; when the paralysis affects only the leg, the lesion is seated in the striate body and its associated fibers.... Finally, in cases in which the hemiplegia is complete, the striate body and the optic layer or their radiations are equally affected. From these observations we conclude that the optic layer and its medullary fibers corresponded to movements of the arm, and the striate body and its radiations to the movements of the leg.10

Foville’s became the standard model of human motor function and it held sway for another 3 decades until Fritch and Hitzig demonstrated the motor function of the cerebral cortex.

Vivisection

Gustav Fritch and Eduard Hitzig’s observation11 that electrocortical stimulation of the gyrus sigmoidalis caused a dog’s limb to move did not convince clinicians that the same applied to humans, especially given that observations performed by Fritch and Hitzig, Hitzig,12 David Ferrier,13 and others were often discordant14 (Table 1). The first dissonant note was struck by Eugene Dupuy, a student of Charles-Édouard Brown-Séquard, who repeated Ferrier’s experiment but was unable to reproduce his results.15 Ferrier, he concluded, had incorrectly assigned voluntary movements to the cortex. Rather, Dupuy thought that the cortex initiated the will to move and that the movements themselves originated in the spinal cord. Others thought that the current applied to the cortex spread from the surface to the basal ganglia, from which movements were generated.16

Charcot dismissed neither Hitzig’s nor Ferrier’s results obtained in animals but thought that they were irrelevant to the human brain, because their brains are so different that it cannot be said, even approximately, which are the corresponding points in one and in the other.... The results obtained by experiments performed in animals, however informative they may be for that species, should not be applied without verification to human pathology.... Therefore, we will need in man, by observation, what Hitzig and Ferrier have done in the monkey and the dog. [To do so, Charcot affirmed, it is] essential to compare symptoms observed in patients to the lesions revealed at autopsy.17

Thus, Charcot turned to the clinicopathological method to determine the cortical localization of human motor function.

The Human Motor Zone

Charcot and Pitres (Figs. 1 and 2) approached the question of localization of what they referred to as the “motor zone” (la zone motrice) in humans by first analyzing a number of patients who had not been paralyzed despite extensive lesions of the frontal lobe in front of the precentral gyrus, of the parietal lobe behind the postcentral gyrus, of the temporal and occipital lobes, and of the insula. From these cases they concluded “that large expanses of the cortical mantle of the cerebral hemispheres have no influence on the performance of voluntary movements.”18 They then hypothesized that “The human motor zone includes...the frontal and parietal ascending convolutions, the paracentral lobule, and probably also the portions of the cortical mantle that is in contact with those parts, such as the feet of the frontal convolutions and the superior and inferior parietal lobules.”19 They found support for this hypothesis in a number of hemiplegic patients in whom the motor zone was observed to be damaged at autopsy but whose basal ganglia were intact.

Charcot and Pitres then turned to patients whose paralysis had been restricted to certain muscle groups while sparing others, to which they referred as partial hemiplegias or associated monoplegias. This included patients whose facial movements were intact, but whose arm and leg had been paralyzed, and others whose lower face and arm had been paralyzed but whose leg was unaffected. From these cases, Charcot and Pitres arrived at a first conclusion about the somatotopic organization of the motor zone:

When destructive lesions occupy a small, specific area of the

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fritch &amp; Hitzig, 1870</td>
<td>Simulation of the gyrus sigmoidalis in the dog produces motor responses of the opposite limb.</td>
</tr>
<tr>
<td>Ferrier, 1873</td>
<td>Repeats Fritch &amp; Hitzig’s experiment in dogs &amp; other mammals.</td>
</tr>
<tr>
<td>Ferrier, 1874</td>
<td>Observes motor responses in primates.</td>
</tr>
<tr>
<td>Charcot, 1875</td>
<td>Results of stimulation obtained in animals cannot be accurately transposed onto the human brain. Begins a clinicopathological study of motor function.</td>
</tr>
<tr>
<td>Ferrier, 1876</td>
<td>Publishes a drawing of the human brain onto which are transposed the results of stimulation obtained in monkeys: motor function extends well beyond the peri-rolandic region, &amp; the somatotopy is incorrect.</td>
</tr>
<tr>
<td>Charcot &amp; Pitres, 1877</td>
<td>Localize human motor function to the peri-rolandic region &amp; establish its correct somatotopy.</td>
</tr>
<tr>
<td>Ferrier, 1878</td>
<td>Dedicates his Gulstonian Lecture to Charcot, bases his discussion of human motor function &amp; somatotopy on Charcot &amp; Pitres’ observations, &amp; reproduces many illustrations from their papers in his lecture.</td>
</tr>
<tr>
<td>Charcot &amp; Pitres, 1879</td>
<td>Publish the first human motor brain map based on autopsies.</td>
</tr>
<tr>
<td>Macewen, 1885</td>
<td>Refers to Charcot &amp; Pitres’ observation on localization to resect a cerebral mass lesion.</td>
</tr>
<tr>
<td>Bennett &amp; Godlee, 1884</td>
<td>Ferrier, aware of Charcot &amp; Pitres’ observations, advises Godlee on his resection of a brain tumor.</td>
</tr>
<tr>
<td>Osler, 1884</td>
<td>Refers to Charcot &amp; Pitres’ localization of the leg area.</td>
</tr>
<tr>
<td>Osler, 1885</td>
<td>Refers to Charcot &amp; Pitres on the resection of epileptogenic brain tumors.</td>
</tr>
<tr>
<td>Jackson, in Bennett &amp; Godlee, 1885</td>
<td>Refers to Charcot &amp; Pitres on the resection of epileptogenic brain tumors.</td>
</tr>
<tr>
<td>Horsley, 1887</td>
<td>Resects brain tumors localized by Jackson.</td>
</tr>
</tbody>
</table>
motor zone, they give rise to partial hemiplegia of the opposite side of the body. Paralysis in these cases do not simultaneously affect the face, the upper limb, and the lower limb. They spare one of these parts and affect the other two. This type of partial hemiplegia (associated monoplegias) is seen in the clinic in two forms. In the first type, the two limbs of the opposite side of the body are paralyzed and the face is spared. In the second type, the lower half of the face and the upper limb on the same side are paralyzed, but the lower limb is not. The site of the lesions is different for each of the two forms. Paralyses of both limbs, without concomitant paralysis of the face result from lesions of the upper part of the motor zone. Associated monoplegias of the face and upper limb result from lesions of the lower motor zone.

Their view of the somatotopic organization of motor function became more specific as they analyzed more complex cases.

Hemiplegic Epilepsy

Charcot and Pitres refined their views on the segmental organization of motor function from their analysis of what they named “hemiplegic epilepsy.” By this term they meant seizures whose “most common characteristics are to begin in an isolated group of muscles and to gradually propagate to the other muscles of the limb and even to the whole body,” and in which there is “paralysis of the affected side in the interval between attacks [for] a few moments, a few hours, a whole day, or, ultimately, becomes true [permanent] paralysis.”

Charcot and Pitres divided the motor zone into thirds. In those patients whose seizure began in the face, lesions were found in the posteriormost part the third frontal convolution and in the inferior and middle thirds of the precentral gyrus. In patients whose seizures began in the arm, lesions were present in the posteriormost aspects of the first and second frontal gyri, and in the middle third of the precentral gyrus. Charcot showed great insight in interpreting these data:

Paralysis from a cortical lesion reflects a suppression of activity: A center is destroyed, the parts that it governs are paralyzed.... On the contrary, convulsions are indicative of abnormal excitation of the centers corresponding to the convulsing parts. The lesion does not act through the center that it has destroyed but because it irritates a region around it.

Charcot was explicit in the significance of this observation: “When, in the intervals between attacks, the patient subject to epileptiform convulsions presents no sort of paralytic phenomena, then the lesion is in the vicinity of the motor zone of the cortex.” This statement encompasses the significance of Charcot and Pitres’ contribution to the nascent field of intracranial surgery: the determination of localization and operability.

Somatotopy

The analysis of their cases led Charcot and Pitres to
Charcot and Pitres also supported their localization by noting that partial seizures that began in the arm were always associated with lesions close to the middle third of the precentral gyrus, and that epileptic attacks in patients whose arm was paralyzed always originated from a lesion in that area. Similarly, Charcot and Pitres found that in some cases in which one side of the face was weak, the patients had difficulty in protruding the tongue, and that it deviated in the direction of the paralysis. They concluded that the lower third of the pre- and postcentral gyri and the foot of the first frontal convolution subserve movements of the tongue.

**Charcot and Pitres’ Brain Map**

From a detailed analysis of more than 98 cases with clinicopathological correlations, Charcot and Pitres concluded, with reference to the accompanying figure (Fig. 3), that

1. On the foot of the third frontal convolution and on the contiguous portion of the ascending frontal convolution were found the center for movements of the tongue.
2. The center for movements of the lower part of the face occupies the two ascending convolutions.
3. The middle third of the ascending frontal convolution includes the center for the isolated movements of the upper limb (the forearm and hand).
4. The paracentral lobule, the upper third of the ascending frontal convolution, and the upper two-thirds of the ascending parietal convolution preside over the two limbs of the opposite side.

This was the first accurate description of human cortical motor areas. It prevailed until Fedor Krause delineated the somatotopy of the precentral gyrus, Harvey Cushing demonstrated the sensory function of the postcentral gyrus, and Otfrid Foerster combined both functions in a single brain map. Wilder Penfield, his colleagues, and his research fellows extended Foerster’s observations by demonstrating the proper orientation of the face, and by delineating the frontal eye field, the second sensory area, the supplementary motor area, and the anterior and posterior speech regions. They identified the role of the amygdala and of hippocampal sclerosis in temporal lobe epilepsy, and with Brenda Milner and Herbert Jasper they localized short-term recall to the hippocampi. In later life Penfield and Jasper integrated the cortex and brainstem as the substrate of mind.

**Discussion**

**Ferrier’s Advocacy**

Ferrier attempted to create a human brain map in 1876 by transposing the results obtained in monkeys onto a sketch of the human brain, but his map was grossly inaccurate: the foot and leg are localized behind the postcentral gyrus; the representation of the arm included the superior frontal gyrus in front of the foot and leg region, the second frontal gyrus in front of the precentral gyrus, and the middle aspect of the precentral gyrus. Furthermore, he localized the mouth to the middle of the precentral gyrus and to the opercular aspect of the postcentral gyri and the foot of the first frontal convolution subserve movements of the tongue.
gyrus, and the lips and tongue to the opercular aspect of the postcentral gyrus. Charcot and Pitres’ brain map, although not as precisely delineated as those obtained with cortical stimulation, had the virtue of being mostly correct.

Ferrier recognized the inadequacy of his human brain map after Charcot and Pitres’ publications. His 1878 Gulstonian Lecture on “The Localisation of Cerebral Disease”29 is dedicated to Charcot, and he relied on Charcot and Pitres’ observations in humans to give relevance to his experiments in animals. Almost all of the illustrations in his lecture are from Charcot and Pitres’ 1877 paper and from those of his students. Thus, Ferrier’s Gulstonian Lecture can be seen as an affirmation of the clinicopathological method and a restating of Charcot and Pitres’ findings on the functional anatomy of the human brain.

Charcot and Macewen

William Macewen, the first surgeon to operate on a brain tumor,30 cited Charcot and Pitres when he related the case of a patient on whom he operated in June 1883 for the resection of a syphilitic gumma, which had first produced weakness of the arm, and then of the leg:

According to Charcot and Pitres cortical cerebral monoplegia affecting the arm and leg coincides with lesions in the superior half of the ascending frontal and parietal convolutions. Viewing the pathological conditions which existed here and the clinical history, it was possible that two separate lesions were present acting coincidently, the one on the arm and the other on the leg. In that case, the lesion affecting the power of the arm would involve chiefly the middle of the ascending frontal and to a less extent the corresponding part of the ascending parietal; while the paracentral lobule would be looked to as the seat of the lesion affecting the lower limb.31

Macewen went on to comment that after Ferrier had visited his wards in 1884, “he soon afterward had the opportunity of advising and assisting Dr. Hughes Bennett and Mr. Rickman Godlee in the case in which the latter removed a tumor from the brain.”32 It seems obvious, therefore, that Charcot and Pitres’ localization of motor function was also instrumental in Bennett and Godlee’s case through Ferrier’s advice. This is not to say that Macewen diminished Ferrier’s contributions, but that he saw them in their proper context:

Localizing symptoms, such as those arising from lesion [sic] in the motor area, have been made use in aiding diagnosis, and in number of instances such symptoms have been the only guide to the cerebral lesion..., for the guides that we have received for the localization of function we are indebted to many observers—Broca, Charcot, Pitres, and Horsley. J. H. Leblanc on the side of patient clinical research, and Hitzig, Ferrier, and others from the purely physiological side, aided by experiments in animals.33

But, as far as localization as an aid to intracranial surgery in patients, he stated that

Abundant proof has been gathered from human pathology, such as that afforded by the elaborate observations of Charcot and Pitres, to put beyond cavil the broad fact that there are points in the human cortex cerebri intimately related to motor and sensory functions of certain parts of the body.34

Charcot, Osler, and Jackson

William Osler also referred to Charcot and Pitres’ observations in a case report in December 1883, before the publication of Bennett and Godlee’s paper.34 Osler’s case was of a girl whose seizures started in the left hand and progressed to the face and leg, with the latter in time becoming paralyzed. Osler found a hard, resistant mass, probably a pilocytic astrocytoma in the superior precentral gyrus. Osler commented on this case, “the tumor occupied...the region which has been found affected in the few recorded instances of paralysis of one lower extremity of cerebral origin. The leg-center is placed in this lobe by Ferrier and Charcot,”34 and restated Charcot’s argument that “as far as man is concerned, while admitting the great and corroborative value of observations upon dogs and monkeys, the careful study of pathological cases offers the only means whereby positive knowledge can be obtained.”35 And, echoing Charcot and Pitres, he concluded, “as far as man is concerned, the analysis of cases would appear to place the leg center in the upper part of the central convolutions...the arm and hand center in the mid-region of the central gyri, and the centers for the face and tongue at the lower end.” As far as operability was concerned Osler again turned to Charcot and Pitres: “Charcot lays down the following rule for guidance in this matter: when in the intervals of the attacks the patient has not any form of permanent paralysis, the disease causing the convulsions is in the non-motor zone, but when, on the contrary, the patient is paralysed in the intervals...we may conclude that there is a destructive lesion in the motor area.” As far as his own case was concerned, Osler concluded that it was “an instance in which an operation would have been justifiable and possibly have been the means of saving life.”36

John Hughlings Jackson, commenting on Bennett and Godlee’s case, also restated Charcot and Pitres with regard to localization of destructive lesions in patients with focal seizures: “Whilst these seizures [point] were with certainty to disease of the cerebral opposite hemisphere they do not always occur from such gross disease as tumour [sic]...so that repeating in effect what Charcot and Pitres had urged, we require also some local persisting paralysis of the part convulsed.”37

Thus, Charcot and Pitres had a determining influence on the first surgeons to breach the dura and on the two most influential proponents of brain surgery.

Through Ferrier’s Gulstonian Lecture, British neurologists were made aware of Charcot and Pitres’ localization of the human motor zone and of its somatotopic organization. By 1886, Victor Horsley had operated on 10 patients, some referred by Jackson and, it can reasonably be assumed, guided by Charcot and Pitres.38 Charcot attended the 1886 meeting of the British Medical Association and related the following to his students in his Tuesday lectures at la Salpêtrière:

Two years ago, at a Congress in Brighton, I held a tumor in my hand that Mr. Horsley had removed from the brain of a man who was also present. He still had a few symptoms of epilepsy, but he was rid of the partial epilepsy from which he had suffered. Mr. Horsley had trephined, sought the tumor, and removed it. It is an extremely simple operation.... There-
fore, we should not worry whenever we are faced with a brain tumor. We must think of the trephine. Someday everyone will do so.39

Conclusions

Neurosurgery evolved from elevating skull fractures and evacuating blood clots from the epidural space to a specialized discipline through the discovery of the structure-function relationship of the human brain in the latter half of the 19th century. Animal experimentation was informative, at least with regard to the canine brain, but the findings were not always concordant and their relevance to humans was unknown. Thus, without reliable information on the site subserving motor function, intracranial surgery was at a standstill. With Charcot and Pitres’ discovery of the human sensorimotor area and its somatotopic representation, brain tumors and epileptogenic scars could be reliably localized and safely resected. This was a major advance in 19th-century brain surgery that laid the groundwork for neurosurgery as it is practiced today.

References


15. Dupuy E. Examen de quelques points de la physiologie du cerveau. Adrien Delahaye; 1873.


29. Ferrier D. The Localisation of Cerebral Disease. Smith, Elder; 1878.


38. Horsley V. Remarks on ten consecutive cases of operations upon the brain and cranial cavity to illustrate the details and safety of the method employed. Br Med J. 1887;1(1373): 863–1865.


Disclosures

The author reports no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Correspondence

Richard Leblanc: Montreal Neurological Institute, Montréal, QC, Canada. richard.leblanc@mcgill.ca.